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Abstract: PURPOSE: It is a widely accepted premise in the scientific community and by athletes alike, that adding resistance exercise to a regular regimen of endurance training increases endurance performance in endurance-trained men. However, critical power (CP), capillarization, and myofiber size remain unaffected by this addition. Therefore, we tested whether the superimposition of resistance exercise with whole-body vibration and vascular occlusion (vibroX) would improve these variables in endurance-trained males relative to resistance exercise alone. METHODS: Twenty-one young, endurance-trained males were randomly assigned either to a vibroX (n = 11) or resistance (n = 10) training group. Both groups trained in a progressive mode twice a week for 8 weeks. Pre and post training, histochemical muscle characteristics, thigh muscle size, endurance and strength parameters were determined. RESULTS: vibroX increased CP (P = 0.001), overall capillary-to-fiber ratio (P = 0.001) and thigh lean mass (P < 0.001), while these parameters were unaffected by resistance training. The gain in CP by vibroX was positively correlated with the gain in capillarization (R (2) = 0.605, P = 0.008), and the gain in thigh lean mass was paralleled by increases in MyHC-1 and MyHC-2 fiber cross-sectional areas and strength. Maximum voluntary torque and the finite work capacity above CP (W') increased significantly only following resistance training. CONCLUSIONS: We achieved a proof of concept by demonstrating that modification of resistance exercise by superimposing side-alternating whole-body vibration and sustained vascular occlusion induced further improvements in CP, capillarization and hypertrophy, all of which were not observed with resistance training alone.

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Abstract

Purpose: It is a widely-accepted premise in the scientific community and by athletes alike, that adding resistance exercise to a regular regimen of endurance training increases endurance performance in endurance-trained men. However, critical power (CP), capillarization, and myofiber size remain unaffected by this addition. Therefore, we tested whether the superimposition of resistance exercise with whole-body vibration and vascular occlusion (vibroX) would improve these variables in endurance-trained males relative to resistance exercise alone. **Methods:** Twenty-one young, endurance-trained males were randomly assigned either to a vibroX (n = 11) or resistance (n = 10) training group. Both groups trained in a progressive mode twice a week for 8 weeks. Pre and post training, histochemical muscle characteristics, thigh muscle size, endurance and strength parameters were determined. **Results:** vibroX increased CP (P = 0.001), overall capillary-to-fiber ratio (P = 0.001) and thigh lean mass (P < 0.001), while these parameters were unaffected by resistance training. The gain in CP by vibroX was positively correlated with the gain in capillarization (R^2 = 0.605, P = 0.008), and the gain in thigh lean mass was paralleled by increases in MyHC-1 and MyHC-2 fiber cross-sectional areas and strength. Maximum voluntary torque and the finite work capacity above CP (W') increased significantly only following resistance training. **Conclusions:** We achieved a proof of concept by demonstrating that modification of resistance exercise by superimposing side-alternating whole-body vibration and sustained vascular occlusion induced further improvements in CP, capillarization and hypertrophy, all of which were not observed with resistance training alone.

Key words: concurrent training, hypertrophy, endurance performance, side-alternating whole-body vibration.

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Introduction

Training regimens of endurance athletes typically consist of both long, continuous, low-intensity exercise and shorter high-intensity interval trainings (HIT), which are designed to improve central (*e.g.* cardiac output) and peripheral (*e.g.* arterial-venous oxygen difference) components (Daussin *et al.* 2007). Taken together, these adaptations increase several parameters of aerobic function, such as critical power (CP), maximal O_2 consumption ($\dot{V}O_{2max}$), gas exchange threshold (GET, "anaerobic threshold"), and exercise efficiency.

It is widely accepted that adding resistance exercise to a regular regimen of endurance exercise in trained athletes brings further positive effects on endurance performance, as first described by Hickson *et al.* (1988). For the sake of clarity, concurrent endurance and resistance training (RT) in this context means that endurance athletes perform their regular weekly endurance training regimen, and add 2-3 sessions of RT per week to their individual training routine (note that recovery time between resistance exercise sessions ≥48 h). The exact temporal sequence of endurance and RT sessions usually is unknown. It was first suggested that the positive effects of resistance exercise on endurance performance are based on alterations in motor unit recruitment (Hickson *et al.* 1988). More recently, however, Kubo *et al.* (2012) proposed that improvements in neural function and increases in tendon stiffness might also represent possible explanations. Despite the findings indicating that endurance performance is improved when regular endurance training is supplemented with resistance exercise, it appears that CP is not influenced by the addition of resistance exercise to the regimen (Bishop and Jenkins 1996). In accordance with the first mentioned aspect, experts agree that it is beneficial for most endurance-trained individuals to supplement their endurance training routine with resistance exercise, as it promotes substantial benefits for the individual, *e.g.* cycling economy (Rønnestad *et al.* 2011).

Despite these benefits, there is at least one issue that should be considered. According to the interference effect (Hickson 1980), endurance exercise has a negative impact on adaptations to resistance exercise, especially on gains in lean mass. This mechanism seems to appear only in young and trained individuals. On a molecular level, it is suggested that 5' adenosine monophosphate-activated protein kinase (AMPK) phosphorylates and activates tuberous sclerosis protein 2 (TSC2), which inhibits the mammalian target of rapamycin complex 1 (mTORC1). The downregulation of the key component in integrating resistance training-activated signaling pathways (mTOR) inhibits the myofibrillar protein synthesis (Atherton *et al.* 2005, Inoki *et al.* 2003). In most cases, the interference effect is even positive for endurance athletes, because they can improve their endurance performance ability without gaining weight, which has to be carried for the competition distance. However, there are cases in which endurance athletes may wish to gain lean mass (*e.g.* road cyclists want to improve their time trial performance or cross country skiers want to improve their sprint performance). The addition of resistance training to their high volume of endurance training leads mostly not to the desired training effect. In these cases, as a result of the high endurance stimulus AMPK is upregulated and hence, the myofibrillar protein synthesis is reduced or inhibited (Inoki *et al.* 2003). These athletes would profit from a novel training modality that allows both a gain in lean body mass and an increase in endurance performance.

Furthermore, it is well accepted that −especially in the case of trained endurance athletes− RT generally neither increases $\dot{V}O_{2max}$, nor mitochondrial content nor capillarization (Aagaard *et al.* 2011, Hickson *et al.* 1988). To further advance endurance athlete performance there is a need for new, effective, and time-efficient training stimuli that are capable of providing endurance performance benefits from resistance exercise, and which will serve to further increase (otherwise possibly capped) structural and functional endurance-type adaptations,

which, in turn, affect CP.

We have recently described a novel training modality termed "vibroX", which might be able to accommodate these needs mentioned above (Item *et al.* 2011, 2013). It simultaneously combines heavy resistance exercise, side-alternating whole-body vibration, and sustained vascular occlusion. In untrained females (Item *et al.* 2011), vibroX elicits a concomitant increase in muscle fiber cross-sectional area (CSA), overall capillary-to-fiber ratio, thigh lean mass, and endurance capacity. Additionally, we found that compared to resistance exercise *per se*, vibroX acutely activates angiogenic and metabolic gene programs, which are normally activated after endurance but not resistance exercise in recreationally trained men (Item *et al.* 2013). Altogether, these results point to a unique role of vibroX in simultaneously mediating endurance- and resistance-type adaptations in untrained or resistance-trained individuals, and thus vibroX has the potential of being considered to be a "total conditioning" stimulus. Moreover, this unique training regimen has been found to be extremely time efficient. In our first study, concurrent adaptations were achieved with a muscle contraction time of approximately 9 min per week for a total of 8 weeks [time commitment per training session: 12-15 min; (Item *et al.* 2011)]. Although we have already shown the benefits of vibroX in young healthy untrained women and young healthy recreationally trained men, a final proof of concept demonstrating that vibroX also works in trained endurance athletes is warranted.

Thus, in this study, we aimed to test whether 8 weeks of vibroX would simultaneously improve CP*,* capillarization, and myofiber size in young, trained male endurance athletes matched for CP at baseline. We chose CP as the functional endpoint of endurance performance, since this approach is believed to be functionally more valuable than the sole

measurement of discrete physiological values such as GET and $\dot{V}O_{2max}$ (Jones *et al.* 2010, Whipp and Ward 2009) or apparently distinct entities such as "short-term and long-term endurance capacity". Another reason for choosing CP as the primary outcome variable was that supplementation of endurance exercise with classical resistance exercise is not believed to impact this variable (Bishop and Jenkins 1996). Based on our previous results (Item *et al.* 2011, 2013) and comprehensive work on the effects of resistance exercise in athletes, ranging from well-trained to highly-trained athletes levels of skill and performance (Aagaard and Andersen 2010, Hickson *et al.* 1988, Rønnestad *et al.* 2012), we hypothesized that only vibroX would increase CP, overall capillary-to-fiber ratio, myofiber CSAs and thigh lean mass simultaneously.

Methods

Participants

Twenty-six endurance-trained males volunteered to participate in this study. The participants were recruited from different cycling, triathlon and academic sport clubs. Two participants aborted the study because of illness or personal reasons. After the pretests, participants were assigned pairwise, according to their CP·kg^{-1} body mass, to a vibroX or RT group. None of the participants had been involved in structured RT prior to the study, and the participants had no or little experience in squat exercise. Study participants were all involved in their early preparation phase of training (pre-season). They were instructed to maintain their individual training routine relating to training frequency, as well as training intensity, and were advised not to include new or additional high-intensity exercise during the study period. Three participants were excluded from the final analysis because they contravened our instruction not to stop their individual training during the study period. Thus, the resulting number of participants was n = 11 and n = 10 for the vibroX and RT group, respectively.

There were no statistical significant differences in physical and performance characteristics between the vibroX and the RT group before training (Tab. 1). Participants were fully informed about the purposes, benefits and risks associated with this study and completed a routine health questionnaire before giving written informed consent to their participation in this study. This study was approved by the ethics committee of the canton Zurich and was conducted in accordance with the declaration of Helsinki.

Experimental procedures

The study consisted of pre- and post-tests for all participants and 8 weeks of either vibroX or RT (Fig. 1). On the first testing day, a percutaneous muscle biopsy was obtained from the *M. vastus lateralis* using a 6-mm Bergström needle (Dixons Surgical Instruments, Essex, UK) as previously described (Item *et al.* 2013). On the second testing day, participants completed an incremental ramp cycle ergometer test to determine peak power (P_{peak}) and peak oxygen uptake ($\dot{V}O_{2peak}$). On that occasion, seat and handle bar of the cycle ergometer (Ergoselect 200 K, Ergoline, Bitz, Germany) were adjusted. These settings were adopted for all of the consecutive trials. After a 3 min rest, the ramp test started at 100 W and involved power increases of 9 W every 18 s (30 W·min^{-1}) until volitional exhaustion. The participants were free to choose the cadence but the chosen revolutions per min (rpm) had to be maintained throughout all the following cycling tests. Volitional exhaustion, *i.e.* task failure, for all cycling tests was defined as the point in time when participants stopped pedaling or the cadence fell more than 5 rpm for > 5 s. On the following testing days, constant-load cycle ergometer tests at 85, 90, 95 and 105% P_{peak} [modified from Brickley *et al*. (2007) by including a 85% stage] were completed in a randomized order to determine CP (Hill 1993, Moritani *et al.* 1981). After a 3 min rest, participants started with a 5-min warm-up at 75 W (Brickley *et al.* 2007). The power was then increased immediately to the given power output.

These endurance capacity tests were conducted until volitional exhaustion as defined above, whereupon time to exhaustion (t_{lim}) was recorded. CP and the finite work capacity above CP (W') were calculated by applying the linear power-time^{-1} equation ($P = W' \cdot t^{-1} + CP$, Hill 1993). The coefficient of determination R^2 for CP was 0.98 ± 0.01 and 0.99 ± 0.02 for the pre- and posttests, respectively. The typical error expressed as a coefficient of variation for CP determined in our laboratory was 3.8%. The familiarization sessions were composed of three attempts for knee extension (dynamometry) and three attempts for both countermovement jumps (CMJ) and squat jumps (SJ). Furthermore, the participants performed two sets of six repetitions of squatting without additional weight. Particular attention was paid to exercise anatomy, form and movement speed.

Equipment and measurements

Cycling tests. During all cycling tests, participants were equipped with a facemask, which covered their mouth and nose (Hans Rudolph, Shawnee, KS, USA). The facemask was connected with an anti-bacterial filter (PALL PRO1087, Pall, East Hills, NY, USA) to an Innocor™ device (Innocor™, Innovision, Odense, Denmark). Pulmonary gas exchange and ventilation were continuously measured breath by breath throughout all ergometer trials. $\dot{V}O_{2peak}$ was determined as the highest mean over a 10-s period.

Knee extension, one repetition maximum (1RM) and jumping test. Knee extensor maximal voluntary torque (MVT) and rate of force development (RFD) were tested using a commercially available dynamometer (Con-Trex® MJ, Physiomed Elektromedizin, Schnaittach/Laipersdorf, Germany). After the body of the participants was stabilized with straps and handles, they performed 3 maximal knee extensions (ω = 3.14 rad·s^{-1}) separated by 1 min to assess MVT. Subsequently, the lever arm of the dynamometer was fixed at a knee angle of 110° (full extension = 180°) and the participants were advised to extend their

leg as fast and as powerful as possible. RFD was calculated for two time intervals: 0-30 and 0-200 ms. Onset of muscle contraction was defined as baseline + 7.5 N·m (Aagaard *et al.* 2002). Squat 1RM measurement was performed according to Niewiadomski *et al.* (2008) over the individual's maximal range of motion. Briefly, the warm-up protocol entailed 8 repetitions at 50% of estimated 1RM and 3 repetitions at 70% of estimated 1RM with a 3 min rest interval between sets. Subsequently, starting with the estimated 1RM, maximal attempts were made to determine 1RM with a 1 min rest interval between attempts (Matuszak *et al.* 2003). The load was successively increased to the point where participants were not able to successfully execute a squat. For the determination of jumping power during CMJs and SJs, three vertical jumps (separated by 30 s of rest) were performed per jumping maneuver on a Leonardo Mechanograph® force plate (Novotec Medical, Pforzheim, Germany). For the detection, storage and analysis of data, we used the manufacturer's software (Leonardo Mechanography GRFD version 4.4, Novotec, Pforzheim, Germany). CMJs were performed with freely moving arms, whereas the SJs were performed with the hands resting on the waist.

Body composition. A densitometer (Lunar iDXA™, GE Healthcare, Madison, WI, USA) was used for the determination of body composition and thigh lean mass. The delineation in region of interest for the thigh was done manually using the integrated software (encore, GE Healthcare, Madison, WI, USA; version 11.40.004) as follows: ROI upper boundary = horizontal line just below the ischium, ROI lower boundary = horizontal line between femur and tibia, ROI lateral boundaries = outer leg cuts.

Muscle biopsy analysis

Tissue sections were cut at 8-μm thickness in a cryostat maintained at −25 °C, and mounted on Fisherbrand Superfrost/Plus microscope slides (Fisher Scientific, Pittsburgh, PA, USA).

The serial cryocut cross-sections were stained using the myofibrillar adenosintriphosphatase (mATPase) method as previously described (Item *et al.* 2011). At least 350 muscle fibers were classified according to their myosin heavy chain (MyHC) isoform into MyHC-1, MyHC-2A and MyHC-2X. For the analysis of oxidative and glycolytic enzyme activities, we incubated the sections in media containing succinate dehydrogenase (SDH) and glycerol-3-phosphate dehydrogenase (GPDH), respectively. At least 350 muscle fibers were counted for each of these two staining procedures. As marker for muscle capillaries, the monoclonal mouse anti-human CD31 endothelial antibody (DAKO, Carpinteria, Canada, 1:600 dilution) was used. Overall capillary-to-fiber ratio was calculated by dividing the number of CD31-positive cells by the number of muscle fibers. At least 50 muscle fibers were counted for the analysis of the capillary-to-fiber ratio (McCall *et al.* 1998, Porter *et al.* 2002). We used the NIH Image J Software (version 1.44o, National Institutes of Health, Bethesda, MD, USA) for all fiber analyses. Fiber CSAs were determined by fully encircling the borders of the mATPase stained cells with Adobe Photoshop Pro CS6 (Adobe Systems Incorporated, San Jose, California, USA) of at least 50 fibers per MyHC isoform (McCall *et al.* 1998). Fiber circularity was calculated using the formula $(4\pi \cdot CSA)/(perimeter)^2$ and only fibers with a circularity higher than 0.7 were considered for analysis (perfect circle = 1.0). Because of the low number of counted MyHC-2X fibers, CSA data were pooled to the 2 major fiber types (MyHC-1 and MyHC-2).

Training regimen

The participants reported twice per week to the laboratory for the supervised training sessions (Fig. 1). The vibroX training consisted of loaded (Multipower®, Technogym, Gambettola, Italy) parallel back squat exercise with superimposed whole-body vibration and sustained vascular occlusion, as described previously (Item *et al.* 2011, 2013). Briefly, squats were

performed on a Galileo® vibration plate (Novotec, Pforzheim, Germany) oscillating at 30 Hz while tourniquet cuffs (0.09 m width, 0.76 m length; VBM, Sulz a.N., Germany) inflated to 200 mmHg (26.7 kPa) were affixed to the inguinal fold region of the thigh. The suprasystolic pressure employed here was the highest pressure that was tolerated by the participants in this setting. One duty cycle consisted of squats until volitional exhaustion, 3 min resting with the pressure of the cuffs maintained, and 1 min resting with cuffs deflated to 100 mmHg (13.3 kPa). RT was conducted analogous to vibroX but without whole-body vibration and vascular occlusion. The participants in the RT group were given a break of 3 min between exercise sets [according to the American College of Sports Medicine (2009)]. Load was set to 70% 1RM at the beginning of the training period and was reduced by approximately 10 % for each of the following sets to induce volitional muscle failure within 60-100 s of exercise. The load magnitude was adjusted progressively during the training period to maintain time under tension between 60 and 100 s (Item *et al.* 2013, Toigo and Boutellier 2006). During the training period, the participants performed two duty cycles or sets of the respective training during the first 4 weeks and three cycles or sets for the remaining of the training period. The technical exercise execution of the squats consisted of a 4 s eccentric action, a smooth 1-2 s transition phase and a 4 s concentric action. After each training, the participants were given a protein shake containing 20 g of whey protein (Nutriathletic Muscle Growth Formula, Scientifics, Schwyz, Switzerland).

Statistical analysis

Data are presented as mean values ± standard deviations (SD). Normality of data was visually ascertained by Q-Q-plots. For the detection of significant differences between groups over time, a univariate general linear model was applied. For this analysis, the differences post−pre (Δ) of each variable was compared between the groups. Significant differences

within groups from pre to post intervention were displayed by parameter estimates. This analysis tested the null hypothesis that Δ parameter was 0. If Δ parameter had a *P*-value lower than the level of significance, the null hypothesis was rejected meaning that Δ parameter was significantly different from 0. A linear regression analysis was used to determine which training-induced improvement best predicted the increase in CP. Pearson correlations were performed to assess the relationships between the variables. The muscle biopsies of two participants could not be analyzed for the mATPase, SDH and GPDH stainings as well as the CSAs due to technical failures. The level of significance was set at $P < 0.05$. All statistical analyses were performed using the software SPSS Statistics 20.0 (SPSS, Chicago, IL, USA).

Results

Starting from a non-significant higher absolute baseline value (Tab. 1), CP increased by 7.9 ± 7.5 W in the vibroX group, while it remained unchanged in the RT group (Fig. 2). Overall capillary-to-fiber ratio increased by 8.2 ± 6.8% in the vibroX group, while the RT group showed no change (−1.0 ± 7.2%, $P = 0.528$), resulting in a significant group x time interaction (Fig. 3a). The increase in overall capillary-to-fiber ratio was the only predictor for the change in CP (adjusted $R^2 = 0.605$, $P = 0.008$) in the vibroX group. Thigh lean mass increased by 3.1 ± 1.7% in the vibroX group, whereas a slight, non-significant increase was observed in the RT group (+1.2 ± 2.0%, $P = 0.074$; Fig. 3b), yielding a significant group x time interaction. MyHC-1 and MyHC-2 CSA's were increased by 24.7 ± 19.9% and 22.2 ± 16.5%, respectively, in the vibroX group, but did not significantly change in the RT group (+3.6 ± 28.2%, $P = 0.643$ and +14.9 ± 29.1%, $P = 0.119$ for MyHC-1 and MyHC-2 CSA, respectively; Fig. 3c, 3d), with no differences between the groups. There was a correlation between the increase in overall capillary-to-fiber ratio and the gain in thigh lean mass in the vibroX group ($R^2 = 0.301$, $P = 0.010$; results not shown). Furthermore, the change in MyHC-

1 CSA correlated with the change in thigh lean mass in the vibroX group (R^2 = 0.631, P = 0.006; results not shown) but not the RT group. MyHC-2A fiber proportion increased in the vibroX group, and also tended to increase in the RT group (Tab. 2). MyHC-1 fiber and MyHC-2X fiber proportions did not significantly change from pre- to post-training in both groups. There were no effects of the two training interventions on GPDH and SDH activity (Tab. 2).

P_{peak} increased by 5.9 ± 6.1% in the vibroX group, while there was a non-significant increase of 2.7 ± 5.9% in the RT group (pre *vs.* post: vibroX: 383.9 ± 45.8 *vs.* 405.2 ± 41.9 W; resistance: 379.9 ± 44.0 *vs*. 389.8 ± 47.4 W). $\dot{V}O_{2peak}$ (pre *vs.* post: vibroX: 4.58 ± 0.54 *vs.* 4.63 ± 0.59 l·min^{-1}, P = 0.136; resistance: 4.16 ± 0.46 *vs.* 4.22 ± 0.45 l·min^{-1}, P = 0.162) and relative $\dot{V}O_{2peak}$ (pre *vs.* post: vibroX: 59.0 ± 6.2 *vs.* 58.9 ± 6.2 ml·min^{-1}·kg^{-1}, P = 0.874; resistance: 55.2 ± 4.2 *vs.* 55.9 ± 4.5 ml·min^{-1}·kg^{-1}, P = 0.156) were not altered in response to the two training interventions. No testing group x time interaction was detected for $\dot{V}O_{2peak}$ (P = 0.983).

1RM increased in both training groups (vibroX: +22.2 ± 10.6%, resistance: +31.8 ± 13.4%; Fig. 4a), with a tendency towards a higher increase in the RT group (P = 0.057). MVT increased in both groups (vibroX: +3.1 ± 8.5%, resistance: +4.9 ± 4.6%) but the increase was significant in the RT group only (Fig. 4b). SJ relative maximum power remained unchanged in the vibroX group while it significantly increased in the RT group (+2.0 ± 4.8% *vs.* +4.7 ± 5.9%, respectively; Fig. 4c). CMJ relative maximum power significantly increased in the vibroX training group by 5.0 ± 8.3%, while the increase did not reach statistical significance in the RT group (+3.1 ± 3.7%; Fig. 4d). RFD during the first 30 ms remained unaffected by both training interventions (P = 0.867 and P = 0.091 for the vibroX and RT group,

respectively; Fig. 4e). RFD during the first 200 ms remained constant for vibroX (+3.0 ± 6.8%, $P = 0.563$) but increased by 14.0 ± 19.5% in response to RT (Fig. 4f). No significant group differences were found for all these variables. W' was not significantly elevated from pre to post vibroX (+5.1 ± 9.6%; pre *vs.* post: 17.8 ± 4.2 *vs.* 18.5 ± 3.7 kJ) while it increased by 10.5 ± 14.9% (pre *vs.* post: 16.7 ± 3.8 *vs.* 18.2 ± 3.5 kJ) in the RT group. No testing group x time interaction was detected for W' ($P = 0.406$). During the first 4 weeks, average time under tension per training session was 135 ± 18 s and 161 ± 6 s in the vibroX and RT group, respectively ($P < 0.001$). In the remaining 4 weeks, average time under tension per training session was 188 ± 17 s and 218 ± 10 s for the vibroX and RT group ($P < 0.001$).

Discussion

This study provides evidence that in trained endurance athletes undergoing regular pre-season training, the addition of vibroX increased CP, capillary-to-fiber ratio and thigh lean mass. Conversely, in the RT group, all these parameters remained unaffected by the addition of conventional RT. The gain in CP was positively correlated with the gain in capillarization, and the gain in thigh lean mass was paralleled by increases in MyHC-1 and MyHC-2 fiber CSAs and strength. The present data extend our previous findings (Item *et al.* 2011, 2013) to trained endurance athletes and thus establish the final proof of concept that in young healthy individuals it is possible to modify a resistance exercise stimulus through superimposition of vibration and sustained vascular occlusion to mediate adaptations otherwise known to occur with endurance exercise only.

In this study, vibroX but not conventional RT, induced muscle hypertrophy, as shown by the specific increases in MyHC-1 and MyHC-2 fiber CSAs and thigh lean mass. Our finding that supplementation of endurance exercise with vibroX promoted muscle hypertrophy is

intriguing, since in young men, adaptations to RT are usually negatively affected by concurrent endurance exercise, *i.e.* simultaneous endurance exercise impairs "strength" and muscle size gains compared with resistance exercise alone (Hickson 1980, Nader 2006) − a phenomenon known as concurrent training or interference effect. Similarly, when endurance exercise is supplemented with resistance exercise in young male trained endurance athletes, typically no increase in muscle fiber CSA and leg lean mass is observed (Aagaard *et al.* 2011, Hickson *et al.* 1988). Here, we clearly showed that vibroX is capable of overcoming this interference effect. It further appears that muscle hypertrophy in the vibroX group was primarily driven by the increase in MyHC-1 fiber size, since we found a positive correlation between MyHC-1 fiber hypertrophy and the increase in thigh lean mass. This finding is not surprising, given the fact that MyHC-1 fiber proportion in the *M. vastus lateralis* of our participants was clearly dominant over MyHC-2 fiber proportions. Conversely, we did not expect that MyHC-1 fibers in trained endurance athletes would undergo further hypertrophy as a result of vibroX training, because these fibers are innervated by small motoneurons with low recruitment thresholds and thus, they are believed to be activated during endurance-type exercise. Remarkably, the advantage of the vibroX training group in relation to muscle fiber hypertrophy was achieved with a significant lower time under tension compared to the RT group. However, average time under tension per training session was, for both training groups, in the predetermined range of 60 to 100 s per set. Therefore, we assume that this difference in training volume had no effect on the outcome. The reduction in time under tension irrespective of the applied load was possibly due to the ischemic pain.

The blunted hypertrophy in response to conventional RT with concurrent endurance exercise might potentially be explained by conditions causing significant cellular energy stress (such as may be the case during high-volume and/or high-intensity endurance exercise), which will

promote an increased activity of 5′-AMP activated protein kinase (AMPK). AMPK is a heterotrimeric serine/threonine kinase, composed of a catalytic subunit (α) and two regulatory subunits (β and γ) (Steinberg and Kemp 2009). It serves as an energy sensor and is activated by an elevation in the AMP/ATP ratio, *i.e.* enhanced binding of AMP to the γ subunit leads to an increase in the phosphorylation of the Thr172 residue on AMPK, and subsequently induces an increase in AMPK activity (Steinberg and Kemp 2009). It has been shown that activation of AMPK inhibits energy consuming anabolic processes such as protein synthesis, and stimulates catabolic energy producing processes such as protein degradation (Steinberg and Kemp 2009). Taken together, there is evidence suggesting that AMPK may negatively affect skeletal muscle mass accretion in the setting of concurrent training by blunting the increase in protein synthesis and increasing protein degradation (Goodman *et al.* 2011).

Contrary to conventional RT, vibroX produced a robust hypertrophic response, which might theoretically either indicate that the addition of vibration and occlusion inhibited AMPK interference or that alternate pathways mediating muscle hypertrophy were activated. An inhibition of AMPK interference might represent a potential mechanism. However, we are not aware of any study investigating this potential mechanism. In contrast, we previously showed, using the same training protocols, that vibroX strongly induces peroxisome proliferator-activated receptor-γ coactivator 1α (PGC-1α) without concomitantly increasing the canonical PGC-1α downstream targets mediating mitochondrial biogenesis in recreationally trained men (Item *et al.* 2013). Interestingly, Ruas *et al.* (2012) have recently identified a form of PGC-1α (PGC-1α4) that results from alternative promoter usage and splicing of the primary transcript. Although it is highly expressed in exercised muscle, it does not regulate most known PGC-1α targets such as the mitochondrial OXPHOS genes. This is in principle consistent with our previous observation (Item *et al.* 2013). Ruas *et al.* (2012)

showed *in vitro* and *in vivo* that PGC-1α4 specifically induces insulin-like growth factor 1 (IGF1) and represses myostatin in the context of resistance exercise, resulting in muscle hypertrophy. In the light of the findings that vibroX specifically and acutely increases PGC-1α mRNA abundance and induces muscle hypertrophy despite concurrent endurance exercise, more research is needed to elucidate the possible roles of PGC-1α splice variants in mediating muscle size adaptation during vibroX.

As shown by linear regression analysis, we found a positive correlation between the increases in CP and capillarization after vibroX training. Notably, these increases occurred without concomitant elevations in $\dot{V}O_{2peak}$ and/or skeletal muscle oxidative enzyme activities. These findings are in line with the notion that in endurance-trained muscles, the augmented capillarization serves other purposes than the oxygenation of tissue, namely a better uptake of substrates and/or removal of fatigue-related metabolites and heat (Saltin *et al.* 1986). Consequently, the enhanced CP observed following vibroX training may, in part, have been the result of better conditions for the uptake and utilization of blood-borne substrates and/or removal of fatigue-related metabolites. Interestingly, it also appears that the increased size of the capillary-to-fiber interface after vibroX was functionally (in terms of CP) more relevant than a decreased diffusion distance consequent to increased capillary density. The reason for this is that vibroX also induced myofiber hypertrophy.

Recently, we showed in recreationally resistance trained young men that an acute bout of vibroX does not increase hypoxia-inducible factor-1α (HIF-1α) mRNA abundance nor the expression of HIF-1α target genes (*e.g.* HIFα prolyl hydroxylase domain 3 [PHD3], lactate dehydrogenase A [LDHA], or phosphofructokinase [PFK]), in spite of a marked increase in the expression of vascular endothelial growth factor (VEGF), PGC-1α, and oxidative stress

markers (Item *et al.* 2013). Given the known sensitivity of PGC-1α signaling to reactive oxygen species (Kang *et al.* 2009, St-Pierre *et al.* 2006), we speculate that VEGF expression was induced in a HIF-1 independent manner, possibly through oxidative stress dependent activation of PGC-1α. Our present finding that vibroX significantly increased capillarization in trained endurance athletes provides converging evidence that vibroX promotes capillarization through non-canonical signaling pathways. The reason being, that in trained endurance athletes most adaptations in terms of capillarization presumably have already occurred, and no further significant effects are to be expected by their classical training routine. Unlike vibroX, RT alone was unable to increase the capillary-to-fiber ratio in endurance-trained individuals participating in this study. This finding is in accordance with a recently published study by Aagaard *et al.* (2011), showing that capillary-to-fiber ratio did not increase following 16 weeks of endurance training supplemented with resistance exercise in elite cyclists.

The increase in W' was only statistically significant in the RT group and twofold greater than in the vibroX training group. This was surprising, because an increase in thigh lean mass, which is believed to influence W' (Miura *et al.* 2002), was observed solely in the vibroX group. Furthermore, the share of MyHC-2A fibers, which is also believed to influence W', increased in the vibroX training group and tended to increase in the RT group at the expense of MyHC-2X and MyHC-1 fibers, even though both decreases were not statistically significant. However, the increase in W' in the RT group was paralleled by increases in MVT and RFD. Therefore, the augmentation in W' was probably due to improved neuronal function. The discrepancy in adaptation between the two training groups might be explained by a ceiling effect. There is a balance between CP and W', both parameters representing distinct parts of energy supply (oxidative and glycolytic, respectively). In an individual

athlete, it is not possible to increase both parameters limitlessly (*e.g.* a marathon runner won't achieve the same *W'* as a 100 m sprinter and *vice versa* for CP). The idea of a ceiling effect might be supported by our findings that *W'*, MVT, and RFD at the beginning of the study tended to be lower in the RT group as compared to the vibroX group but were not different any more after the exercise period. The notion that a ceiling effect exists is further supported by the results of Bishop and Jenkins (1996). In their study, untrained males exhibit similar gains in 1RM, comparable to our RT group (+28.6% *vs.* +30.0%, respectively), but the magnitude of the increase in *W'* was markedly higher (+34.9% *vs.* +6.5%, respectively). The above-mentioned slight increase in MyHC-2A fiber proportion might have benefited the overall outcome of the cycling tests, but presumably only to a small extent. This finding is in line with the results of Aagaard *et al.* (2011), who showed that even in elite competitive cyclists, proportional distribution of MyHC-2A increases after a RT intervention. Similar to the results of previous studies investigating the effects of additional RT in endurance-trained athletes (Aagaard *et al.* 2011, Hickson *et al.* 1988, Losnegard *et al.* 2011), RT alone increased 1RM, MVT, RFD and SJ relative maximum power in this study, demonstrating that our RT protocol was a valid and effective control condition.

While we demonstrated that vibroX is a potent new training stimulus, we can only speculate about some of the mechanisms, which may mediate its effects. Specifically, the role and contribution of the single vibroX components are still unknown. On the one hand, the compressive cuff during vascular occlusion restricts arterial inflow, resulting in hypoxia and greater metabolic acidosis, while concomitantly blocking venous outflow resulting in continual stimulation of the afferents (Manini and Clark 2009). Blood flow restricted exercise thus may result in greater stimulation of chemosensitive sensory nerves arising from the active musculature (namely class III and IV afferents), which in turn result in an acute

increase in serum growth hormone and catecholamines. In addition, β-adrenergic receptor density is approximately threefold greater in type 1 than in type 2 fibers (Martin *et al.* 1989, Martin *et al.* 1992). Together with the possibility that β-adrenergic stimulation might induce a PGC-1α signaling (possibly through PGC-1α4) this might constitute a hypothetical mechanism, which could help to explain why type 1 fiber hypertrophy occurred during vibroX training. On the other hand, we speculate that the vibration-induced reflex activity was associated with the recruitment of distinct, task-related subpopulations of motor units ("functional motor unit pools") (Burke 2002). There are indeed examples in which two or more functional motor unit pools share a single muscle compartment, and that the motoneurons of these functional pools are intermixed within the same spinal motor nucleus (Burke 2002). In theory, vibration-induced reflexes during vibroX could therefore lead to task-specific recruitment of additional motor units that otherwise are not recruited during squat exercise. It is clear, however, that adaptations to vibroX are significantly more (in terms of quantity and quality) than the sum of the adaptations usually observed for its single components, and these gains may be achieved with a minimal training time commitment. Moreover, given that vibroX produces universal (contrary to specific) effects, further research is warranted to elucidate the mechanisms underlying adaptations/effects and to establish optimal dose-response relationships. In addendum, we would like to point out that, until now, vibroX was only applied in healthy, untrained to trained participants and that the safety in other groups of participants, especially patients, has not been evaluated, yet.

Conclusions

In conclusion, this study has provided the final proof of concept that modification of resistance exercise by superimposing side-alternating whole body vibration and sustained vascular occlusion induced further improvements in CP, capillarization and hypertrophy in

trained endurance athletes, all of which had not been observed with RT alone. The increment in CP was positively correlated with the increase in overall capillary-to-fiber ratio. The gain in thigh lean mass was mainly due to MyHC-1 fiber hypertrophy. Increases in W' and dynamometry parameters were only detected after RT, while both groups showed improvements in 1RM and maximum jumping power. On the basis of these findings, we recommend that trained endurance athletes who aim at further improving their endurance performance should consider vibroX as an additional new training modality.

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Table 1. Physical characteristics and pre training values for the vibroX and resistance training (RT) groups.

	vibroX (n = 11)	RT (n = 10)
Age [y]	26.7 ± 3.5	28.4 ± 4.8
Height [cm]	181.5 ± 6.6	178.8 ± 6.3
Body mass [kg]	77.9 ± 8.1	75.5 ± 7.0
CP [W]	286.4 ± 37.8	274.3 ± 43.9
CP·kg^{-1} [W·kg^{-1}]	3.70 ± 0.53	3.65 ± 0.62
1RM [kg]	128.1 ± 20.2	130.3 ± 39.2
MVT [Nm]	169.4 ± 24.8	166.5 ± 29.5
RFD 30 ms [Nm·s^{-1}]	1447.8 ± 526.5	1272.8 ± 405.6
RFD 200 ms [Nm·s^{-1}]	1088.9 ± 280.0	978.7 ± 280.1
SJ $P_{max,rel}$ [W·kg^{-1}]	49.4 ± 4.0	51.6 ± 6.0
CMJ $P_{max,rel}$ [W·kg^{-1}]	55.2 ± 6.4	60.2 ± 6.0
Overall CTF ratio	1.82 ± 0.20	1.86 ± 0.19
MyHC-1 fiber CSA [10^{-9} m^2]	4.51 ± 0.66	4.82 ± 1.16
MyHC-2 fiber CSA [10^{-9} m^2]	5.78 ± 0.67	6.33 ± 1.03
Thigh lean mass [kg]	13.6 ± 1.9	13.4 ± 1.2

Values are mean ± SD: CP, critical power; 1RM, one-repetition maximum; MVT, maximal voluntary torque; RFD, rate of force development; SJ, squat jump; CMJ, countermovement jump; $P_{max,rel}$, relative maximal power; CTF, capillary-to-fiber; MyHC, myosin heavy chain; CSA,

Table 2. Distribution of the myosin heavy chain (MyHC) types, glycerol-3-phosphate dehydrogenase (GPDH) activity, and succinat dehydrogenase (SDH) activity pre and post training period in the vibroX and resistance training (RT) group.

	vibroX (n = 9)		RT (n =10)	
	Pre	Post	Pre	Post
MyHC-1	58.7% ± 6.7%	56.6% ± 7.0%	59.2% ± 14.7%	56.1% ± 11.7%
MyHC-2A	36.6% ± 8.6%	40.7% ± 5.6%*	37.3% ± 13.7%	42.3% ± 12.1%†
MyHC-2X	4.7% ± 4.3%	2.7% ± 3.3%	3.5% ± 3.1%	1.6% ± 1.7%
GPDH activity	47.9% ± 9.4%	50.3% ± 9.4%	44.3% ± 13.4%	48.2% ± 12.3%
SDH high activity	60.9% ± 6.0%	60.9% ± 5.3%	61.7% ± 7.1%	60.4% ± 8.3%
SDH weak activity	38.5% ± 5.9%	38.2% ± 5.3%	37.7% ± 7.1%	39.3% ± 8.5%
SDH no activity	0.7% ± 1.1%	1.0% ± 2.2%	0.7% ± 0.9%	0.3% ± 0.5%

Values are mean ± SD. *$P < 0.05$; †$P = 0.051$ pre *vs*. post.

Legends

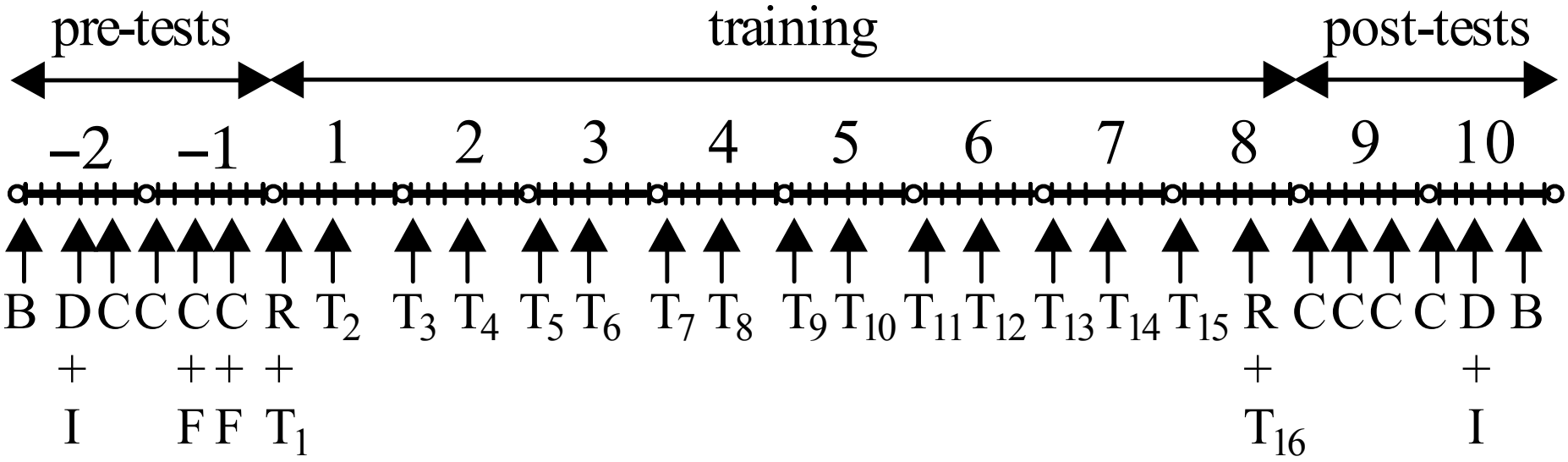
Figure 1: Study design on a weekly scale (−2 to 10). B, biopsy; C, constant-load tests; D, dual-energy x-ray absorptiometry; F, familiarization; I, incremental ramp test; R, testing of 1RM, dynamometry and jumping mechanography; $T_{1\text{-}16}$, training sessions.

Figure 2: Critical Power (CP) pre (white bars) and post (black bars) training in the vibroX and resistance training group. Bars and error bars represent mean values and SD, respectively, for 21 participants (vibroX: $n = 11$, resistance $n = 10$). $^{**}P < 0.01$, significant differences within group pre *vs.* post; $^{\#}P < 0.05$, significant differences between groups pre *vs.* post.

Figure 3: a) Overall capillary-to-fiber ratio, b) thigh lean mass, c) myosin heavy chain type 1 (MyHC-1) fiber cross-sectional area (CSA), and d) MyHC-2 fiber CSA pre (white bars) and post (black bars) training in the vibroX and resistance training group. Bars and error bars represent mean values and SD, respectively, for 21 (Fig. 3a, 3b; vibroX: $n = 11$, resistance $n = 10$) or 19 participants (Fig. 3c, 3d; vibroX: $n = 9$, resistance $n = 10$). $^{*}P < 0.05$, $^{**}P < 0.01$, significant differences within group pre *vs.* post; $^{\#}P < 0.05$, $^{\#\#}P < 0.01$, significant differences between groups pre *vs.* post.

Figure 4: a) One repetition maximum (1RM), b) maximum voluntary torque (MVT), c) squat jump (SJ) relative maximum power ($P_{max,rel}$), d) countermovement jump (CMJ) relative maximum power ($P_{max,rel}$), e) rate of force development (RFD) during the first 30ms of contraction, and f) RFD during the first 200ms of contraction pre (white bars) and post (black bars) training in the vibroX and resistance training group. Bars and error bars represent mean values and SD, respectively, for 21 participants (vibroX: $n = 11$, resistance $n = 10$). $^{*}P <$

1 0.05, ***$P < 0.001$, significant differences within group pre *vs.* post.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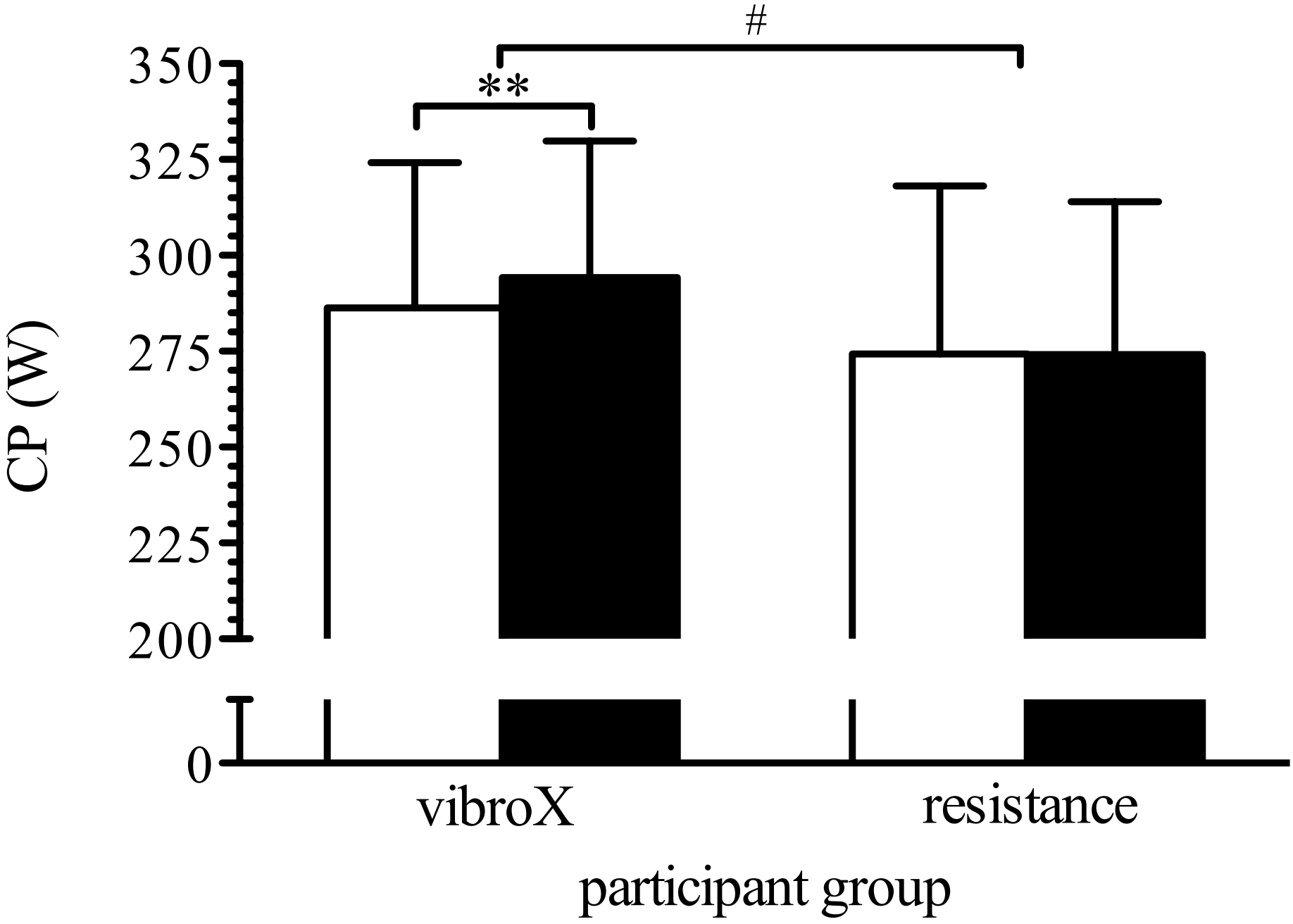
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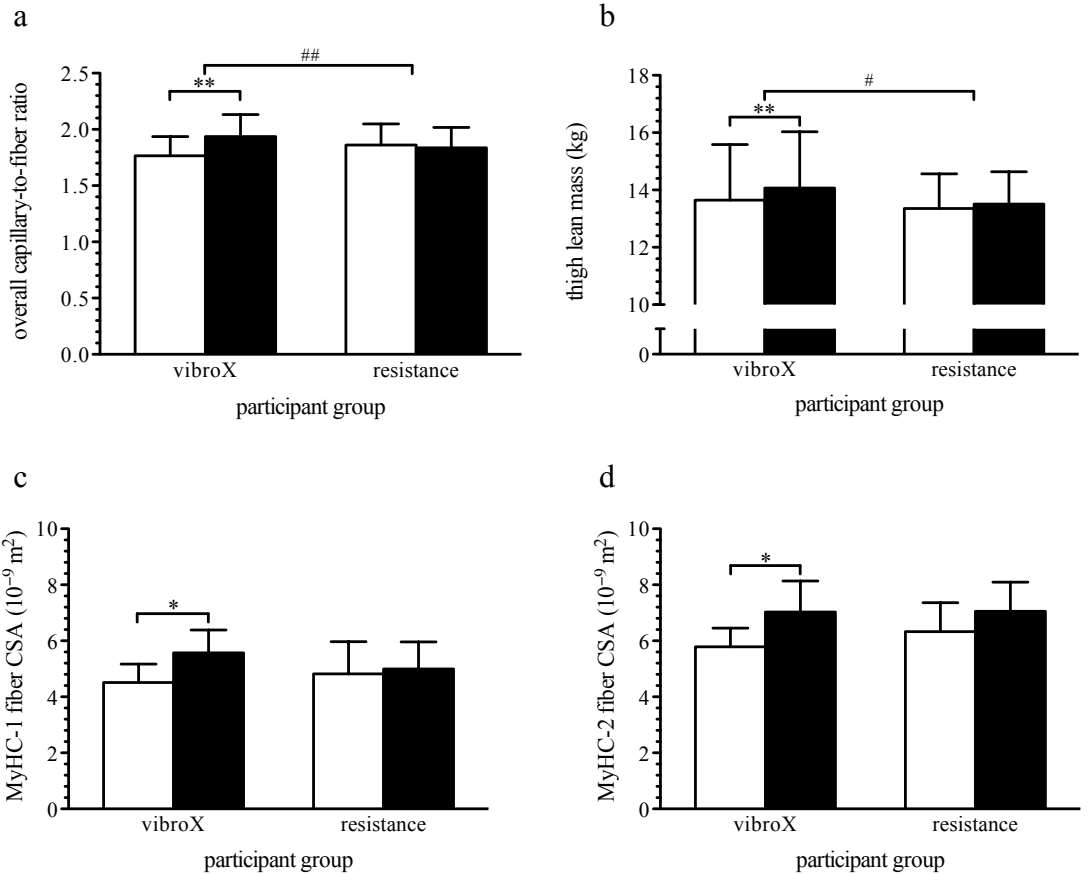
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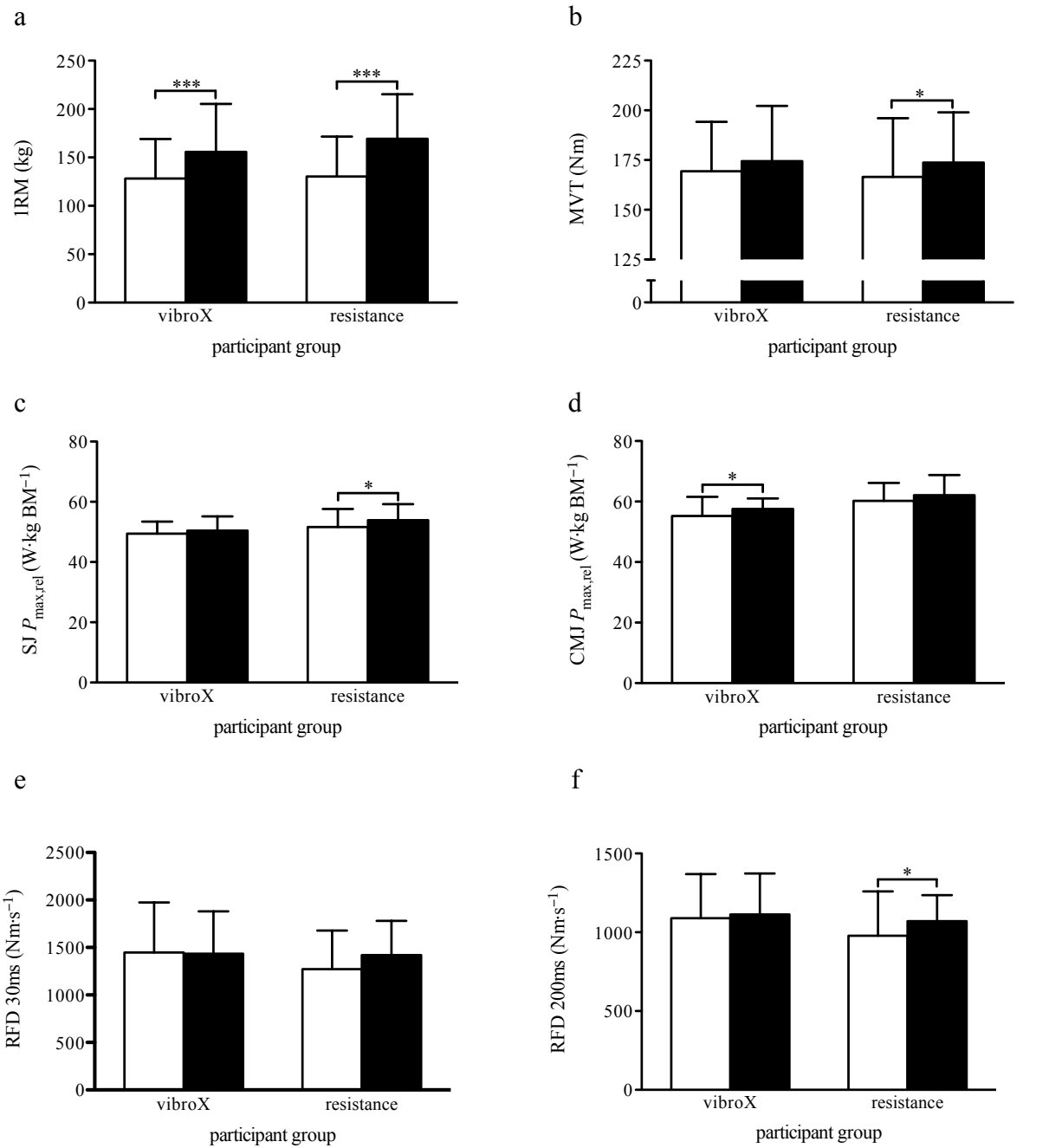
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